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CHAPTER

19

**Early Surgical Management of Poor-Grade
Patients with Intracranial Aneurysms**

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INTRODUCTION

Most surgical series dealing with treatment of cerebral aneurysms concentrate on patients who are in good neurological condition. In contrast, the natural history of patients with poor neurologic grades (grades III-V) indicates that the 6-month mortality is high. For example, 50% of grade III patients on admission will be dead within 6 months. For patients of grades IV and V, 65-95% will be dead within 6 months. The majority of these patients will die as a result of either their initial hemorrhage or acute rebleeding episodes or vasospasm. Recently, there has been increased experience and interest in early surgery for patients with aneurysmal subarachnoid hemorrhage (3, 5, 6, 8). However, most of these reports tend to concentrate on patients who are in good neurological condition. Because of increased experience in early surgery and the high mortality in poor-grade patients managed conservatively, we hypothesized that early surgery combined with aggressive postoperative management could improve on the natural history of poor-grade aneurysmal patients.

Patients and Methods

Beginning in January 1984, we instituted a policy of early surgical (<72 hours) intervention in all patients with subarachnoid hemorrhage (SAH) admitted to Harborview Medical Center. Harborview Medical Center is the major emergency facility for Seattle and has frequent referrals from Washington, Alaska, Montana, and Idaho. A highly organized prehospital emergency medical system exists which concentrates medical care of trauma, burns, and other catastrophic disorders, such as SAH and stroke. All patients with SAH admitted through our Emergency Department were seen by the Neurosurgery Service. If necessary, aggressive resuscitative measures were insti-

tuted either at the prehospital site (*i.e.*, in the field) or the emergency room. In the field, intubation and fluid resuscitation were standardly utilized. After stabilization in the emergency room, immediate unenhanced computed tomography (CT) scanning was followed by infusion CT. Infusion CT scanning consisted of fine-cut CT scans through the circle of Willis during a constant infusion of contrast material. In a previous study, this method was shown to detect 97% of aneurysms greater than 5 mm in diameter (10). In most cases, immediate, four-vessel angiography was then obtained. Surgery was performed as soon as possible in all cases depending on operating room and personnel availability.

One hundred seventy-two patients with non-giant anterior circulation aneurysms, which had ruptured, were admitted to Harborview Medical Center between January 1, 1984 and April 1, 1988. All patients sustained an SAH documented by lumbar puncture or CT analysis. Sixty-one of these 172 patients were admitted more than 72 hours after their SAH and are excluded from this analysis. Twelve remaining patients had no brainstem function despite aggressive resuscitative measures in the emergency room and were diagnosed as having a ruptured anterior circulation aneurysm by CT or postmortem examination. These 12 patients were thus brain dead and were therefore not considered in our analysis. The remaining 110 patients represent all anterior circulation aneurysm patients admitted to Harborview Medical Center with partial or full brainstem function. No patient was excluded from surgery as long as partial brainstem function existed. Thus, a case management analysis would include all 110 patients whom we operated on within 72 hours of their hemorrhage. Preoperatively, patients were graded according to Nishioka (11).

The mean age of our patients was 51 years with a range of 16–83 years. There were 69 females and 41 males. The location of the ruptured aneurysms is indicated in Table 19.1. Thirty-seven percent of the patients had an anterior communicating artery aneurysm, 30% had a middle cerebral artery aneurysm, and 33% had an internal carotid artery aneurysm.

The neurologic grade of the patients is indicated in Table 19.2. Seventy-two percent of our patients were in grades III, IV, or V. Because of the unique emergency medical triage system in the Seattle location, we believe the distribution in neurologic grade represents a valid representation of the distribution of patients with acute SAH, with the majority of patients being in poor-grade categories.

Surgery in most cases was performed through a pterional

TABLE 19.1
Characteristics of Population

Age range:	16-83 yr	(mean, 51 yr)
Sex:	69 Females	41 Males
Location of aneurysm	No.	%
ACA ^a	41	37
MCA	33	30
ICA	36	33

^a ACA, anterior cerebral artery; MCA, middle cerebral artery; ICA, internal carotid artery.

approach with lumbar subarachnoid drains and deliberate hypotension. Temporary clipping was rarely utilized. Hematoma evacuation was carried out with aneurysm clipping in almost all cases. Unruptured multiple aneurysms, if located in proximity to the ruptured aneurysm, were obliterated at the same time of clipping of the ruptured aneurysm.

Of the 110 patients, 105 had preoperative angiography. A subset of patients did not undergo angiography but only infusion CT. These 5 patients were in extremis because of large intracerebral hematomas, as diagnosed by CT, and were emergently taken to the operating room for clot removal and aneurysmal clipping. Preoperative infusion CT in these patients correctly identified the location of the aneurysms.

Postoperatively, all patients underwent four-vessel angiography, either on the day of surgery or within 48 hours of surgery. All had intracranial pressure monitoring postoperatively and were cared for in our Neurosurgery Intensive Care Unit. Frequent postoperative CT scans were obtained. All patients underwent preoperative and postoperative fluid loading, and Swann-Ganz catheters were used to allow on-line monitoring of cardiovascular parameters and intermittent determination of cardiac output. Depending on the patient's clinical status, xenon-131 cerebral blood flow, single photon emis-

TABLE 19.2
Neurologic Grade

Grade	No. of Patients	%
I	4	3
II	27	25
III	23	21
IV	34	31
V	22	20

sion tomography, and transcranial Doppler ultrasound (TCD) were utilized to monitor their postoperative course.

CT Characteristics

Data from the Cooperative Aneurysm Study reported by Adams *et al.* (4) showed a definite relationship between initial CT findings and outcome after SAH.

We analyzed the 94 available CT scans for three factors: (a) degree of SAH according to Fisher *et al.* (7); (b) presence of intracerebral hemorrhage (ICH); and (c) presence of hydrocephalus. As indicated in Table 19.3, a large proportion of our patients had either localized clots in vertical layers ≥ 1 mm (31%) or diffuse or no SAH with ICH or intraventricular hemorrhage (IVH) (37%). We separately analyzed for the presence of ICH (Table 19.4). Of the 42 patients with intraparenchymal hemorrhages, 27 had significant midline shifts. An additional 20 patients had IVH.

As indicated in Table 19.5, initial CT scanning revealed infrequent occurrence of acute hydrocephalus of a severe nature (17%). In contrast, 45% of our population had no hydrocephalus, whereas 38% showed moderate hydrocephalus.

Diagnosis of Delayed Ischemia

Patients were carefully observed clinically for the development of symptoms of delayed ischemia. If either a focal neurologic deficit or a significant decrease in level of consciousness developed that could not be explained by other factors (*e.g.*, edema, hemorrhage, electrolyte disturbance, increased intracranial pressure, etc.), patients were subjected to a repeat four-vessel angiogram to check for vaso-

TABLE 19.3
CT Grade^a: 94 Patients

	No. of Patients	%
A (no blood)	2	2
B (diffuse blood, vertical layers <1 mm)	28	30
C (localized clots, vertical layers ≥ 1 mm)	29	31
D (diffuse or no SAH with ICH or IVH)	35	37

^a Grading as described in Reference 7.

TABLE 19.4
Presence of Intracerebral Hemorrhage

	No. of Patients	%
Intraparenchymal hemorrhage	15	16
Without shift	27	29
With shift	20	21
IVH	45	48
SAH		

spasm. Since 1986, TCD ultrasound has also been utilized, and the findings have correlated well with vessel narrowing seen on angiogram.

Treatment of Cerebral Vasospasm

The treatment utilized for ischemic neurologic deficit due to vasospasm mainly consisted of hypervolemic, hypertensive therapy, as outlined by Kassell *et al.* (9). Beginning in the recovery room, all patients had fluid loading. If neurologic deterioration due to vasospasm was diagnosed, isotonic crystalloid solutions, as well as colloid solution in the form of human serum albumin, were administered to keep the pulmonary capillary wedge pressure in the range of 12–18 mm Hg. If the worsened condition persisted, cardiac output and blood pressure were increased with inotropic drugs until the deficits resolved or the cardiac output was 1½–2 times pretherapy value. More recently, we have utilized percutaneous balloon angioplasty to treat deficits secondary to vasospasm.

RESULTS AND OUTCOME

Mortality

The overall mortality measured at 6 months was 25%, with 28 of 110 patients dying during this time period. As indicated in Table 19.6, there was a strong correlation ($P < 0.005$, χ^2 analysis) between neurologic grade on admission and mortality.

TABLE 19.5
Presence of Hydrocephalus

	No. of Patients	%
None	42	45
Moderate	36	38
Severe	16	17

TABLE 19.6
Mortality Related to Neurologic Grade

Grade	No. of Patients	%
I	0/4	0
II	4/27	15
III	3/23	13
IV	12/34	35
V	9/22	41

$P < 0.005$ by $\chi^2 = 8.06$.

CT Correlation with Mortality

The presence or absence of hydrocephalus had no correlation with mortality. In contrast, the amount of blood in the basal cisterns measured by CT grade, according to Fisher *et al.* (7), correlated strongly with mortality. For example, patients having no blood or diffuse blood with vertical layers < 1 mm had less than 4% mortality. In contrast, patients with localized subarachnoid clot ≥ 1 mm had a mortality of 28%, and patients with an ICH or IUH had a mortality of 46% (Table 19.7).

There was also a strong association between mortality and the presence of a midline shift in patients with intraparenchymal hemorrhages (Table 19.8). Fifty-nine percent of patients with intraparenchymal hemorrhage and shift were dead within 6 months, in contrast to only 13% of patients with intraparenchymal hemorrhage without shift. IUH was associated with death in 25% of the patients.

Mortality Related to Location

As indicated in Table 19.9 overall, patients with middle cerebral artery aneurysms have the highest mortality (42%), in contrast to the mortality associated with anterior cerebral (20%) or internal carotid artery aneurysm (17%). This association persisted even

TABLE 19.7
Mortality Related to CT Grade

CT Grade	Mortality	
	No. of Patients	%
A	0/2	0
B	1/28	4
C	8/29	28
D	16/35	46

$P < 0.001$ by $\chi^2 = 14.57$.

TABLE 19.8
Mortality Related to Intracerebral Hemorrhage

	No. of Patients	%
SAH only	6/45	13
Intraparenchymal hemorrhage		
Without shift	2/15 ^a	13
With shift	16/27 ^a	59
IVH	5/20	25

^a $P < 0.005$ by $\chi^2 = 8.3$.

when neurologic grade was considered (Table 19.10). For example, if the better-grade patients (grades I-III) are separated from the poor-grade patients, patients with middle cerebral artery aneurysms still had a higher death rate. For example, in the grade I-III group, patients with middle cerebral aneurysms had a 29% mortality, in contrast with the 6-8% mortality at other locations. In the poor-grade patients with middle cerebral artery aneurysms, mortality was 53%, in contrast to a 35% mortality in patients with anterior communicating artery aneurysms. Poor-grade patients with internal carotid artery aneurysms had a significantly lower (25%) mortality, compared to poor-grade patients with middle cerebral artery aneurysms.

DISCUSSION

We are reporting our experience with early surgery in anterior circulation aneurysm with special emphasis in poor-grade patients. Poor-grade patients represented more than 70% of our aneurysmal hemorrhage population admitted to our Medical Center. Our overall mortality was 25%. There was an increase in mortality which correlated strongly with neurologic grade on admission. Thus, mortality was low (0-13%) with patients admitted in grades I-III. In contrast, grade IV patients had a 35% mortality and grade V patients had 41% mortality. As previously documented (12), the 6-month mortality in

TABLE 19.9
Mortality Related to Location

	No.	%
ACA	8/41	20 ^a
MCA	14/33	42
ICA	6/36	17 ^a

^a $P < 0.05$.

TABLE 19.10
Mortality: Location Related to Clinical Grades

	Grades I-III		Grades IV-V	
	No.	%	No.	%
ACA	2/24	8	6/17	35
MCA	4/14	29	10/19	53
ICA	1/16	6	5/20	25

conservatively treated patients with a single aneurysm SAH was 52%, 65%, and greater than 90% in patients of grades III, IV, and V, respectively. Thus, in comparison to the natural history, acute surgical intervention in poor-grade aneurysm appears to be better than the natural history.

This decreased mortality can be attributed to several factors, such as aggressive resuscitative measures in the prehospital and emergency room phase of the patient's care, rapid availability of CT and angiographic capability, and operative facilities. Comprehensive postoperative management is also of vital importance in these critically ill patients. For example, we aggressively managed hydrocephalus, both pre- and postoperatively. In addition, routine postoperative management included intracranial pressure monitoring and treatment of increased intracranial pressure by either medical or surgical (ventriculostomies) means. Hypervolemic therapy was routinely instituted in all patients and aggressively pursued. Xenon-131 blood flow and single photon emission computerized tomography (SPECT) studies provided information and insights in the management of patients with postoperative cerebral ischemia secondary to vasospasm.

This aggressive postoperative strategy and technique outlined above are not unique. However, we have utilized two additional postoperative modalities which we have found useful and believe will have widespread utility in the future: TCD ultrasound for the diagnosis of vasospasm and balloon angioplasty for the treatment of vasospasm.

TCD ultrasound, introduced by Aaslid in 1982 (2), utilizes a 2-MHz frequency to obtain velocity of blood flow from the arteries at the base of the brain. Normal velocities of blood flow in the middle cerebral arteries range from 30-80 cm/sec (mean, 62 cm/sec). Marked increases in velocity occur with vessel narrowing and correlate to vasospasm seen on angiogram (1). If either severe vessel narrowing

were seen on angiogram or high velocities were seen on TCD indicating significant vasospasm in the face of a neurological deterioration, therapy for vasospasm was initiated.

Percutaneous balloon angioplasty, a technique previously reported by Zubkov *et al.* (13), utilizes a transfemoral angiography approach using a microballoon catheter which can then be directed into the major vessels of the circle of Willis. The balloon is then inflated multiple times in the spastic vessel, dilating it in a manner similar to coronary angioplasty (Fig. 19.1). Ten patients have been treated by this method in this institution. Eight have improved, permanently reversing their neurologic deficits caused by vasospasm.

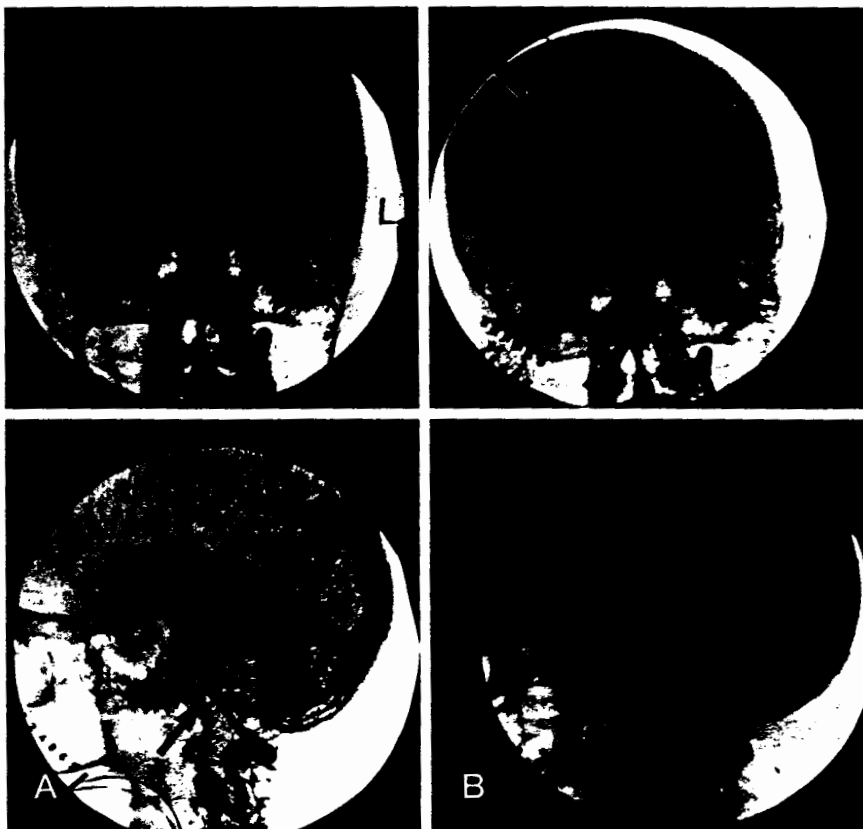


FIG. 19.1. (A) Angiogram showing severe vasospasm of the left distal vertebral artery after rupture of an anterior circulating aneurysm (arrows indicate region of spasm). (B) Angiogram showing the same vessel after dilation and using percutaneous balloon angioplasty (arrows indicate site of dilation).

These encouraging results thus far indicate that this technique may be a valuable tool in the future treatment of vasospasm.

When this series is compared retrospectively to the natural history of SAH in patients who were treated by bed rest, there appears to be an improvement in mortality in poor-grade patients. This improvement in mortality can be attributed to the early surgery plus the aggressive pre- and postoperative management.

REFERENCES

1. Aaslid, R., Huber, P., and Nornes, H. Evaluation of cerebrovascular spasm with transcranial Doppler ultrasound. *J. Neurosurg.*, 60: 37-41, 1984.
2. Aaslid, R., Markwalder, T. M., and Nornes, H. Noninvasive transcranial Doppler ultrasound recording of flow velocity in basal cerebral arteries. *J. Neurosurg.*, 57: 769-774, 1982.
3. Adams, H. P., Kassell, N. F., Kongable, G. A., and Torner, J. C. Intracranial operation within seven days of aneurysmal subarachnoid hemorrhage. Results in 150 patients. *Arch. Neurol.*, 45: 1065-1069, 1988.
4. Adams, H. P., Kassell, N. F., and Torner, J. C. Usefulness of computed tomography in predicting outcome after aneurysmal subarachnoid hemorrhage. A preliminary report of the Cooperative Aneurysm Study. *Neurology*, 35: 1263-1267, 1985.
5. Chyatte, D., Fade, N., and Sundt, T. M. Early *versus* late intracranial aneurysm surgery in subarachnoid hemorrhage. *J. Neurosurg.*, 69: 326-331, 1988.
6. Disney, L., Weir, B., Grace, M., and The Canadian Nimodopine Study Group. Factors influencing the outcome of aneurysm rupture in poor-grade patients: a prospective series. *Neurosurgery*, 23: 1-9, 1988.
7. Fisher, C. M., Kistler, J. P., and Davis, J. M. Relation of cerebral vasospasm to subarachnoid hemorrhage visualized by computerized tomographic scanning. *Neurosurgery*, 6: 1-9, 1980.
8. Gilsbach, J. M., Harders, A. G., Eggert, H. R., and Hornyak, M. E. Early aneurysm surgery: a 7-year clinical practice report. *Acta Neurochir.*, 90: 91-102, 1988.
9. Kassell, N. F., Peerless, S. J., Durward, Q. J., Beck, D. W., Drake, C. G., and Adams, H. P. Treatment of ischemic deficits from cerebral vasospasm with intravascular volume expansion and induced arterial hypertension. *Neurosurgery*, 11: 337-343, 1982.
10. Newell, D. W., LeRoux, P.D., Dacey, R. G., Stimac, G. K., and Winn, H. R.: CT infusion scanning for the detection of cerebral aneurysms. *J Neurosurg* (In Press)
11. Nishioka, H. Evaluation of the conservative management of ruptured intracranial aneurysms. *J. Neurosurg.*, 25: 574-592, 1966.
12. Winn, H. R., Richardson, A. E., and Jane, J. A. The assessment of the natural history of single cerebral aneurysms that have ruptured. In: *Clinical Management of Intracranial Aneurysms*, edited by L. N. Hopkins and D. M. Long, pp. 1-10. Raven Press, New York, 1982.
13. Zubkov, Y. N., Nikiforov, B. M., and Shustin, V. A. Balloon catheter technique for dilatation of constricted cerebral arteries after aneurysmal subarachnoid hemorrhage. *Acta Neurochir.*, 70: 65-79, 1984.